severe. Preceding his entrance to the hospital he attacked an individual on the street while in a state of post-epileptic mania. The opium was administered from March 23d to May 4, then bromides. He had two attacks, not followed, however, by delirium. Is quiet and busy, but depressed at times.

Case 7. Man, age 29, has been epileptic since infancy, was operated on (trephined) in another hospital, but without success. The attacks occur every few days, followed by stupor. On May 13th the opium was begun, and on June 21st the bromides. The attacks are no longer followed by stupor.

The other three cases are on the bromide treatment preceding the opium, and the author does not wish to make any report of their condition as yet. KRAUSS.

A Case of Sporadic Cretinism, Treated With Thyroid Extract.

By L. Haskoree (*Wiener Med. Wochenschrift*, 1895, No. 43 44).

The author does not recognize, under sporadic cretinism, cases in which there is a congenital absence of the thyroid gland. The patient, 38 years of age, was physically well built, while intellectually her abilities to accomplish anything were very discouraging. A goitre had gradually developed since her eight year.

Treatment with thyroid extract for six weeks showed an appreciable, and in fact, marked diminuation in the size of the goitre. The disposition of the patient became one of restless activity, besides being also very irritable. Her powers of comprehension were clearer. As accompanying symptoms there was an increased rapidity of the pulse rate, slight rise of temperature, a copious perspiration, tremor of the upper extremities, and a diminution in the weight of the patient.

MITCHELL.

"Sur la Valeur Hypnotique du Trional Chez Les Enfants."

By Moncorw (*Bull. de l'Acad. de Med.*, 1895, No. 35).

According to this author a very prompt and efficient hypnotic for children is trional. Psychical and nervous excitement is easily controlled, and no bad after effects have been noticed, even though it may be necessary to continue the use of the drug for several days. In small doses (0, 2 —0, 25), a sedative effect can be obtained. MITCHELL.

CLINICAL.

Early or Juvenile General Progressive Paralysis.

By Dr. Alzheimer (*Allgem. Zeitschrift fur Psychiatree* Vol. LII. No. 3, 1895, pp. 533-594).

This long article contains a valuable summary of the thirtyeight cases of juvenile progressive paralysis, hitherto published. The author also puts on record two cases of his own, into the histories of which he enters very minutely. One of these was a girl of illegitimate birth, her father was a syphilitic; the patient herself had symptoms of heredtiary syphilis. At the age of three years, meningitic symptoms? Intelligence medium, memory tardy. At eleven years of age tonic convulsions. She began to menstruate at fifteen, soon after marked diminution of visual acuity, unsteady gait, clumsiness of the hands. At nineteen years of age, St. Vitus' dance and increasing difficulty in walking, blunting of the mental faculties up to marked idiocy, incontinence, decubutus. Admitted to the hospital at twenty-two years of age, undeveloped appearance, marked emaciation, unequal sluggish pupils, tremor of the muscles of the face and of the mouth, paralytic disturbance of speech, reflexes marked by exaggerated, ankle clonus, dementia, exitus letalis seven weeks after admission, paralytic marasmus.

The autopsy, which was made three and a-half hours after death, showed the following: dura mater, adherent; pia mater, cloudy, thickened and adherent; convolutions markedly atrophied, particularly in the frontal and parietal lobes; ventricles greatly dilated; ependyma granular; both thalami apparently hypertrophied; spinal cord does not appear to be macroscopically altered.

Microscopic examination revealed marked degeneration of the lateral tracts of the cord; the posterior columns intact; many ganglionic cells of anterior horn fatty, pigmented and sclerotic; a few in Clarke's column similarly affected; medulla exhibits same changes to slight degree; atrophy nervi optici; in the frontal convolutions, the tangential fibres and the fibres of the the second layer have almost disappeared; numerous sections of different convolutions show complete disappearance of demarcation between the individual cortical layers; blood vessels increased in number, the walls greatly infiltrated; there are few normal ganglionic cells, most of them in advanced sclerotic degeneration; increase in nuclei of neuroglia; intense degeneration of tissue of optic thalami, corpora striata, and of parts of the base of the brain under the ganglia; carmine stained preparations show an extraordinary increase of the glia cells.

Case 2. Girl of legitimate birth, hereditary taint on mother's side, father had ante-nuptial syphilis; patient had probably symptoms of hereditary syphilis; during infancy she had hydrocephalus, and her conduct was abnormal. In school she complained frequently of headaches, tendency to somnolence. In 1886 (nine years old) she had a paralytic attack (forced movements of head, without complete loss of consciousness).

A few days thereafter, weak gait, abstraction ; following that excitability and anxiety ; mental functions diminished. In 1888 two attacks of tonic convulsions, with short periods of unconsciousness, followed by vomiting and hallucinations. In 1889, frequent attacks of considerable diversity of character ; psychical condition worse ; unequal, sluggish pupils ; speech and writing disturbances ; twitching of muscles of face ; patella reflexes exaggerated ; ankle clonus ; disturbances of bladder ; hallucinations of hearing. In 1890, at times confused ; increasing dementia ; spastic contracture of right side. In 1891, increasing contracture of left side. In 1894, admission to hospital ; great dementia ; left side and right lower extremity spastic and contractued ; right arm paretic but not rigid ; death in three weeks.

Autopsy twenty hours post-mortem. The following changes were found : dura adherent ; pia, cloudy, thickened, very œdematous, and adherent to frontal lobe ; convolutions markedly atrophied ; brain substance uncommonly hard ; ventricles greatly dilated ; optic thalami apparently diminished in size, particularly the right thalamus ; on the under surface of the cerebellum, a morbid focus of the size of a ten-cent piece. Spinal cord—cervical part of dura considerably thickened ; lateral tracts discolored a distinct gray. Microscopic examination. Spinal cord—intense sclerosis of both lateral tracts and of left anterior pyramidal tracts ; ganglionic cells in places fatty and pigmented ; the degeneration of the pyramidal tracts can be traced to the peduncles where it disappears ; on the floor of the fourth vertricle and in the nuclei of the medulla are many ganglion cells, with extensive pigmentation ; other cells are granular and cloudy.

Convolutions cerebri—in general, show the usual changes ; sections from different convolutions show remarkable poverty in medullary fibres ; sections hardened in alcohol and stained in chromin show unusual width of the first cortical layer ; the second layer is normal, whilst the fourth and particularly the fifth layer exhibit atrophy, which accounts for the apparent hypertrophy of the first layer ; the ganglion cells show marked degenerative changes ; the cells of the basal ganglia are likewise affected.

Case 3. Girl, illegitimate birth ; mother a prostitute and syphilitic ; now probably a progressive paralytic ; father also a syphilitic. Patient had probably symptoms of hereditary lues. At twenty-one years of age, sight diminished ; admitted to hospital; atrophy of optic nerves ; headaches; lightning-like pains in the back ; absence of patella reflexes. At twenty-six years, periods of excitement ; speech disturbance, fibrillary twitching of tongue and face muscles ; dementia; paralytic attacks; delusions. Opportunity for post-mortem examination not yet presented.

The comparative study of all of these cases of juvenile progressive paralysis may be summarized as follows :

I. PARTICIPATION OF BOTH SEXES.

Of the forty-one cases, twenty were males and twenty-one females, a proportion of 1:1. In progressive paralysis of the adult, the male sex is preponderingly predisposed in the proportion of females as 2–4:1 in cities, and 5–12:1 in country districts.

II. TIME OF ONSET.

AGE.	NO. OF CASES.
9–10	3
11–12	4
13–14	8
15–16	1
17–18	15
19–20	4
21–22	2

This table shows that the affection may appear as early as the ninth year of age. Up to the sixteenth year, there is an increasing predisposition, which diminishes toward those later years when the progressive paralysis of adults begins to appear.

III. DURATION OF THE DISEASE.

The duration of the affection was studied in twenty-three cases as follows :

DURATION.	NO. OF CASES.
2–3 years	5
3–4	4
4–5	5
5–6	3
6–7	1
7–8	3
8–9	2

The average duration is thus shown to be 4.5 years. It is also to be observed that the duration of juvenile progressive paralysis is longer than in the paralysis of the adult.

IV. HEREDITY.

In eleven of the cases an hereditary taint was not elicited. In four cases distinct denial of an hereditary taint is made, whilst in twenty-six cases hereditary predisposition was observed. Including all the cases, there is a percentage of hereditary predisposition of 63.4%. Omitting the eleven cases mentioned, the percentage leaps to 86.6%. In paralysis of the adult the percentage is far less.

V. ETIOLOGY.

Inspection shows at once the important role played by syphilis. In 50% of the cases, syphilis was the positive causative factor. Counting the cases in which the history showed the probable presence of syphilis, the percentage amounts to 85%. Table showing relation of syphilis to Juvenile progressive paralysis:

	GROUPS.		NO. OF CASES.
1.	History of syphilis defective		7
2.	History shows absence of syphilis		3
3.	Direct infection positive		3
4.	Hereditary syphilis	probable	2
		very probable	12
		positive	14

An interesting question in this connection is how many years may elapse before the appearance of paralysis in a subject who has inherited syphilis? The record shows that the disease may make its appearance as late as the twenty-fifth year.

VI. GENERAL INTELLIGENCE BEFORE THR ONSET OF THE DISEASE.

Three cases were weak-minded from infancy; five cases were of limited intelligence; six cases were of medium intelligence; nineteen were of good intelligence; eight cases make no mention of the intelligence. There is, therefore, a considerable number of juvenile paralytics, who from childhood up, show less than average intelligence.

VII. CAUSE OF DISEASE.

A, Mental Symptoms.—In the majority of cases the disease runs its course under the form of simple dementia, without delusions. The absence of other psychical symptoms, particularly delusions of grandeur is striking in juvenile progressive paralysis as compared with the adult type.

C, Physical Symptoms.—The characteristic physical symptoms are: 1. The frequency of the paralytic attacks. 2. The physical paralysis dominated frequently from the beginning, the cause of juvenile progressive paralysis, whilst the mental symptoms are more slow in development. 3. Frequently one side, at times one extremity, is more paralyzed than the other.

The cases can be easily confounded with spastic spinal paralysis, brain tumor, and with multiple sclerosis.

Symptoms pertaining to the pupils, speech, tremor of the muscles of the tongue and face, present nothing of note differing from those in adult paralysis.

In nineteen cases no mention was made of the patella reflex; in seven cases the reflex was absent; in fifteen cases the reflex was exaggerated. Thus in 31.8% there was a combination of paralysis with tabetic symptoms.

The frequent appearance of optic nerve atrophy is worthy of note. It was observed in 12% of the cases. It is relatively less frequent in adults.

The bodily development of juvenile paralytics is very often below the average for the corresponding age in health. The disease appears to hold the physical evolution in check.

VIII. AUTOPSY AND MICROSCOPIC EXAMINATION.

Many observers content themselves with the statement that the post-mortem changes are typical of progressive paralysis. When details are given, thickening, cloudiness, and œdema of the pia, frequent adhesion of the same to the cortex, atrophy of the convolutions, particularly of the frontal lobes, dilatation of the ventricles, ependymitis granulosa, are found. In two cases there was in addition a pachymengitis hemorrhagica. In one case mention is made of a subdural membrane. In another, a localized softening of the temporal lobes. In the case of Bjeljakow osteophytic thickening of the bones of the skull was found. In the case of the author, a focus was discovered in the cerebellum.

The average weight of the brain was 957 gr. whilst normally it is 1,300 gr.

The microscope reveals nothing different from what is found in case of adult progressive paralysis. In both there is degeneration of the medullary fibres, fatty pigmentation and sclerosis of the ganglion cells, degeneration and infiltration of the walls of the vessels and accumulation of glia cells. In one of the author's cases, the atrophy of the deeper cervical layers was in striking contrast to that of the mere superfical layers. This is not found in adults. The author also calls particular attention to the atrophy of the basal ganglia in his cases.

IX. FREQUENCY OF PARALYSIS IN THE PERIOD OF DEVELOPMENT.

In comparison with the adult form, the paralysis à début prècoce is rare. In the institution with which the author is connected, three cases of the juvenile form as against 360 of the adult form, were seen in a period of six years. Elkins (*The Lancet*, 24, 1894), states that in the Edinburgh asylum, from 1889 to 1890, 166 adults and eight juveniles were received. This, the author considers, a surprisingly high percentage.

MEIROWITZ.